

# The Effects of Ambient Ozone on Lung Function in Children: A Reanalysis of Six Summer Camp Studies

Patrick L. Kinney,<sup>1</sup> George D. Thurston,<sup>2</sup> and Mark Raizenne<sup>3</sup>

<sup>1</sup>Division of Environmental Sciences, Columbia University School of Public Health, New York, NY 10032 USA; <sup>2</sup>Institute of Environmental Medicine, New York University Medical Center, Long Meadow Road, Tuxedo, NY 10987 USA; <sup>3</sup>Health Canada, Ottawa, Ontario, Canada

Studies of children attending summer camps often have observed relationships between daily outdoor ozone ( $O_3$ ) concentrations and decreased lung function that are qualitatively similar to results seen in human chamber studies. The former studies, focusing on the pulmonary effects of  $O_3$  and associated pollutants on children under natural conditions of exposure, are potentially of great importance to understanding the public health impact of ambient  $O_3$ . However, a thorough assessment of the results of these studies has been hampered by differences in the analysis and reporting of data across the various studies. We obtained data sets from six summer camp studies carried out by three separate investigative groups, including two New Jersey studies performed by New York University, two studies in Ontario carried out by Health and Welfare Canada, and two studies in southern California. The data consisted of sequential, daily measurements of forced expiratory volume in 1 sec ( $FEV_1$ ), peak expiratory flow rate (PEFR), and 1-hr  $O_3$  concentration in the hour preceding lung function measurements for each child. We analyzed the relationships between lung function and  $O_3$  using linear regression models that fit subject-specific intercepts and a single, pooled  $O_3$  slope. These models were fit for each of the six studies separately and for all studies combined. All of the study-specific slopes of  $FEV_1$  on  $O_3$  were negative (i.e., increased  $O_3$  associated with decreased  $FEV_1$ ); five of six were statistically significant. Analysis of the combined six-study data set yielded a slope of  $-0.50$  ml  $FEV_1$ /ppb  $O_3$  ( $p < 0.0001$ ). Addition of time-trend variables to the combined-data analysis diminished, but did not eliminate, the  $FEV_1$ - $O_3$  relationship. Study-specific slopes for PEFR on  $O_3$  were more variable. Combined over studies, no significant relationship was observed between PEFR and  $O_3$ . However, this negative finding appeared to be partially confounded by time trends in PEFR. The results of this reanalysis provide strong evidence that children exposed to  $O_3$  under natural conditions experience decreases in  $FEV_1$  of the kind demonstrated in laboratory studies, and raise concern that other acute respiratory effects observed in those studies (e.g., pulmonary inflammation) may also occur in young people exposed to ambient  $O_3$ . **Key words:** air pollution, children, epidemiology, lung/pulmonary function, ozone, summer camps. *Environ Health Perspect* 104:170-174 (1996)

Ozone ( $O_3$ ) is a ubiquitous pollutant in outdoor air, frequently reaching 1-hr concentrations that exceed the National Ambient Air Quality Standard of 120 ppb. Because  $O_3$  is formed through photochemical reactions involving nitrogen oxides and hydrocarbons, the highest and most broadly peaked  $O_3$  concentration profiles often occur downwind of, rather than directly in, major source areas such as large cities.

Short-term exposures to ambient-level  $O_3$  concentrations have been shown to result in a spectrum of effects on the human respiratory system, including drops in lung function (i.e., measures of lung volume and expiratory flow rates), increases in lung reactivity to other irritants, and pulmonary inflammation (1-4). The bulk of the data revealing these effects derive from chamber studies, which typically involve the study of relatively small numbers of healthy, white, adult, male volunteers exposed to pure  $O_3$  under controlled laboratory conditions along with some form of exercise. While each individual chamber study has necessari-

ly been limited in scope, the large number of chamber studies carried out in the past 20 years has yielded a rather extensive database for certain outcomes, most notably lung function. McDonnell and colleagues (5) accumulated data on 290 adult, white, male subjects from 5 separate chamber studies, with the goal of determining whether individual lung function response to  $O_3$  is related to age, height, baseline lung function, allergen sensitivity, and other factors. Of these factors, only age had a consistent influence on responsiveness, with response diminishing with increasing age.

From the perspective of assessing the public health impact of  $O_3$  exposures under real-world conditions, however, the chamber study database presents some limitations. First, the precisely controlled exposure conditions are unlike those that occur during many ambient episodes, where broad, multi-hour peaks, often accompanied by fine particulate matter, are observed. Second, relatively few chamber data are available on children and those

with moderately severe lung disease, two population subgroups that may be at elevated risk of ozone-induced health impacts.

Epidemiologic studies have examined the acute pulmonary effects of ambient  $O_3$  under natural conditions. These "field studies" can be categorized into three general types: summer camp studies, exercise studies, and daily life studies. All three designs involve collection of repeated measurements on individuals and analyze the association between lung function and day-to-day changes in  $O_3$  concentrations, with each subject serving as his or her own control. In contrast to chamber studies, field studies examine the human health effects of  $O_3$  under real-world conditions, with natural patterns of  $O_3$  exposure and levels of co-pollutants and other environmental factors.

The most extensively and consistently used field study design for investigating acute  $O_3$  effects has been the camp study, which involves collection of sequential (usually daily) lung function data on children attending summer camps, along with concurrent measurements of air pollution concentrations and meteorological conditions. Camp studies, involving sequential, usually daily, measurements of lung function and air pollution concentrations, offer the significant advantage that individual pollution exposures in a potentially large subject population can be easily and accurately estimated using a single, on-site monitoring station. In addition, these studies directly investigate associations of pulmonary function with natural diurnal patterns of  $O_3$  and associated co-pollutants, which often involve prolonged daytime peaks. Finally, the focus is on children, whose lungs are still developing.

Address correspondence to P. L. Kinney, Division of Environmental Sciences, Columbia University School of Public Health, 60 Haven Avenue, B-1, New York, NY 10032 USA.

We thank Mort Lippmann and Dalia Spektor of New York University for providing the Fairview Lake study data sets; Haluk Ozkaynak of Harvard University for the Pine Springs study data set; and Diane Gibbons of General Motors Corporation, who provided Haluk Ozkaynak with the Pine Springs data. The two Canadian camp studies and the San Bernardino study data sets were provided by Mark Raizenne. Funding was provided by contracts from Health and Welfare Canada.

Received 14 April 1995; accepted 20 November 1995.

Many camp studies have supported the conclusion that ambient  $O_3$  exposures in active children are associated with short-term declines in population average lung function (3,6). Other studies have yielded negative or equivocal results (7–10). It is not clear to what extent these differences are merely due to variations in data analysis and reporting methods across studies and to what extent they may represent substantive differences in results. Concerns about potential confounding by other pollutants or weather variables, which may co-vary with  $O_3$ , have led some to question the attribution of the observed associations to  $O_3$  per se. These uncertainties have limited the causal inferences that can be drawn from the camp studies and have limited their use in risk assessment and regulatory decision making.

Due to the common study design, data from camp studies are amenable to reanalysis. By pooling data from several studies, a more robust overall relationship between naturally occurring  $O_3$  exposures and decreased lung function can be estimated. A collective analysis also can yield insights into differences across studies in the relationship between lung function and  $O_3$ , without the influence of differing analytical methods.

This article presents the results of our reanalysis of the original lung function and  $O_3$  data from six recent summer camp studies. These studies include two in northwestern New Jersey (3,11), two in southern California (6,9), and two in Ontario (8,12). Using a consistent analytical method, results are reported on the relationship between lung function and  $O_3$  for each individual study and for all studies combined.

## Methods

The 1984 Fairview Lake, New Jersey, study was conducted by researchers from New York University and Harvard University during a 4-week period in July and August at a summer YMCA camp in northwestern New Jersey (3). Once per day, usually in the afternoon, lung function was measured on each of up to 91 children (53 males and 38 females, 8–15 years of age). The Fairview Lake study was repeated in 1988, this time with 46 campers (33 males and 13 females, 8–14 years of age) and with function measurements collected in both the morning and the late afternoon (11). This design allowed consideration of changes in function during the course of each day.

The Lake Couchiching study was conducted collaboratively by Health and Welfare Canada and Harvard University over 10 days in late June and early July of

1983 (7,12). Lung function data were collected twice daily (0730–0930 hr and 1630–1830 hr) for 52 campers (including 23 asthmatics: 12 males and 11 females, and 29 nonasthmatics: 16 males and 13 females), who ranged from 7 to 15 years of age. These researchers also conducted a follow-up study at a Girl Guide camp located beside Lake Erie in southern Ontario during 6 weeks in June, July, and August 1986 (7,8). In that study, referred to as “CARES,” spirometry was performed daily on 112 female campers, who averaged 11.6 years of age.

Two summer camp studies were conducted in California with support from General Motors Corporation. The first took place at a church-sponsored summer camp in the San Bernardino Mountains (elevation 1740 m) (6). Over a 3-week period in June and July 1987, 43 children (19 males and 24 females, 7–13 years of age) performed spirometry up to three times per day. During late June through early August 1988, a follow-up study was conducted at Pine Springs Ranch, east of Los Angeles (9,10). Lung function testing was carried out twice daily on each of 295 campers (ages 8–17) who attended one of six separate, sequential, 1-week camps.

Working data sets for each study reviewed above were obtained either directly from members of the original study teams, or through third parties (see Acknowledgments). A common feature of all six data sets was the availability of afternoon spirometric lung function data collected over many days on many subjects, along with the previous 1-hr average  $O_3$  concentrations. For consistency, the present analysis focused on afternoon lung function measurements. Except as noted, if more than one afternoon lung function measurement was available for a given subject on one day, only the last measurement was used in the analysis. Because too few asthmatic subjects were available to analyze as a separate group (only the Lake Couchiching study included asthmatics), asthmatics were excluded from these analyses.

In each of the original studies, lung function data were collected by spirometry using methods that conformed closely to guidelines published by the American Thoracic Society (13). The present analysis was limited to data on forced expiratory volume in 1 sec ( $FEV_1$ ) and peak expiratory flow rate (PEFR). Ozone measurements were collected using automated real-time monitors (based on either UV photometry or chemiluminescence), with instrument checks and calibrations conducted at regular intervals.

Data were analyzed using analysis of covariance methods via the GLM procedure of the SAS system (SAS Institute, Cary, North Carolina). The analysis was performed using data on four variables from each study: subject ID,  $FEV_1$ , PEFR, and the 1-hr average  $O_3$  concentration in the hour preceding the lung function measurements.

For each study, linear regression models were fit relating  $FEV_1$  or PEFR (the dependent variables) to  $O_3$  (the independent variable). Regression models included a single, pooled  $O_3$  slope and separate intercepts for each subject (to account for differences in average lung function across subjects). After obtaining study-specific results, the combined six-study data set was used to estimate the mean slopes across all studies for  $FEV_1$  or PEFR regressed on  $O_3$ . These analyses were repeated with the addition of linear or higher-order functions of test number in order to fit the time-trend in repeated lung function measurements noted in previous studies (9,14,15). Ambient temperature was not included as a covariate in the analysis because human chamber studies have shown that direct effects of temperature on lung function are minimal within the normal ambient range (16).

## Results

Table 1 presents data summaries for the six studies. Tables 2 and 3 present slope estimates for  $FEV_1$  and PEFR, respectively, for each of the six studies analyzed separately. Nearly all of the slopes were negative, indi-

**Table 1.** Key descriptive statistics for six studies of the lung function response of children to air pollution

| Study                  | Total no. of subjects | Total no. of observations | Mean observations/subject | Mean $O_3$ (ppb) <sup>a</sup> | Maximum $O_3$ (ppb) | Mean $FEV_1$ (l) | Mean PEFR (l/sec) |
|------------------------|-----------------------|---------------------------|---------------------------|-------------------------------|---------------------|------------------|-------------------|
| Fairview Lake, 1984    | 91                    | 1237                      | 13.6                      | 53                            | 113                 | 2.14             | 4.36              |
| Fairview Lake, 1988    | 46                    | 577                       | 12.5                      | 69                            | 137                 | 2.39             | NA                |
| Lake Couchiching, 1983 | 29                    | 244                       | 8.4                       | 59                            | 95                  | 2.41             | 5.48              |
| CARES, 1986            | 112                   | 1228                      | 11.0                      | 71                            | 143                 | 2.34             | 5.51              |
| San Bernardino, 1987   | 43                    | 255                       | 5.9                       | 123                           | 245                 | 2.06             | 5.07              |
| Pine Springs, 1988     | 295                   | 1826                      | 6.2                       | 94                            | 161                 | 2.19             | 4.52              |

Abbreviations:  $FEV_1$ , forced expiratory volume in 1 sec; PEFR, peak expiratory flow rate; NA, PEFR data not available for this study.

<sup>a</sup>1-hr average, at time of afternoon lung function measurement.

cating that higher concentrations of  $O_3$  were consistently associated with decreased lung function.  $FEV_1$  slopes spanned an approximate fourfold range (-0.2 to -1.3 ml/ppb). Five of the six slopes were statistically significant. Peak flow results (Table 3) were less consistent. Four of the five slopes were negative, two of which were statistically significant. The PEFR slope for the Pine Springs study was unique in being positive and statistically significant. Individual analysis (not shown) of the 6 separate weeks of the Pine Springs camp revealed that only week 6 had a statistically significant positive slope of PEFR on  $O_3$ . Over the 6 days of data collection during week 6, both PEFR and  $O_3$  increased markedly, resulting in a strong positive, but likely spurious, correlation between these two variables. The increase in PEFR probably reflected the positive training effect that has been noted in previous studies. These results illustrate the potential for confounding of  $O_3$  effects by time trends in repeated spirometry. Exclusion of week 6 from the analysis of Pine Springs data resulted in a slightly positive, but nonsignificant, overall slope for Pine Springs. There was no evidence that the overall results for the Pine Springs camp were unduly influenced by data from a subset of subjects with very narrow ranges in  $O_3$  exposures.

The relationship between lung function and  $O_3$  was analyzed for the combined, six-study data set (Table 4). The overall  $FEV_1$  slope on  $O_3$  was -0.50 ml/ppb (SE = 0.07;  $p$  = 0.0001). The overall PEFR slope was positive but non-significant. This PEFR result was heavily influenced by data from the Pine Springs camp, which, as noted earlier, had a positive slope and had the largest number of subjects (295) and observations (1826). When data from this one study were set aside, the PEFR slope was -0.99 ml/sec/ppb (SE = 0.33;  $p$  = 0.003).

Exploratory analysis of the time trend in  $FEV_1$  (independent of  $O_3$ ) showed that  $FEV_1$  tended to drop over the first four to five measurements, followed by a gradual increase and leveling off (results not shown). This trend was well fit by a third-order polynomial (i.e., linear, squared, and cubed trend variables were all statistically significant in a multiple regression analysis). The temporal pattern for PEFR was adequately fit by a simple linear increase over time. The temporal patterns observed here were qualitatively similar to those reported in a recent study from Holland (15). The regression of lung function on  $O_3$  was repeated with these time-trend variables included in the models (Table 5). The overall  $FEV_1$  slope on  $O_3$  was reduced (in absolute magnitude) by about half in

this model: -0.26 ml/ppb (SE = 0.07;  $p$  = 0.0003). Thus, inclusion of variables accounting for temporal trends in  $FEV_1$  reduced but did not eliminate its relationship with  $O_3$ . The overall PEFR slope was -0.15 ml/sec/ppb (SE = 0.34;  $p$  = 0.65) with the linear time-trend variable in the regression model. Setting aside the Pine Springs data resulted in an overall PEFR slope of -1.06 ml/sec/ppb (SE = 0.33;  $p$  = 0.001). Thus, the PEFR slope on  $O_3$  became slightly more negative after controlling for time trends.

## Discussion

This study analyzed the relationship between daily variations in lung function and ambient  $O_3$  concentrations for children attending six summer camps. When analyzed individually using a common method,  $FEV_1$  was inversely related to  $O_3$  concentrations at each of the camps. Pooling the data across camps, an average  $FEV_1$  decline of 0.5 ml/ppb  $O_3$  was observed. Pooled analysis of PEFR indicated no statistically significant overall relationship with  $O_3$  concentrations. However, there was strong evidence for heterogeneity across camps, with four of five available studies yielding negative slopes (two of which were statistically significant) and one yielding a significant positive slope. The latter result appeared to be confounded by a strong training effect for PEFR.

While negative slopes relating  $FEV_1$  and  $O_3$  were seen for each camp, there was

variation in the slope estimates across camps. This variation was found to be statistically significant using an  $F$ -test for heterogeneity (results not shown). Possible reasons for these differences include camp-to-camp variations in subject activity levels (resulting in different  $O_3$  doses at a given, measured exposure level), differences in temporal patterns of  $O_3$  concentrations (with broad peaks likely to have greater effects on lung function per ppb  $O_3$ ), potentiation of the  $O_3$  effect by other pollutants, variations across populations in inherent  $O_3$  sensitivity and/or prior adaptation to  $O_3$ , and confounding by meteorologic factors or airborne allergens. Because of limitations in the available data, the relative roles of each of these factors in the observed variations across camps can be discussed only in qualitative terms.

There are few quantitative data on subject activity levels at the six camps. Although activity surely contributes to the random variability within and between camps, it cannot be quantified. While  $O_3$  concentration profiles are often sharp and peaked in cities (e.g., Los Angeles), all of the camps analyzed here were located outside of major source areas and exhibited similarly shaped diurnal  $O_3$  patterns, implying that this is unlikely to be an important differential factor. Potentiation of  $O_3$  effects on lung function in asthmatics by acid aerosols has been demonstrated in a chamber study in which  $O_3$  exposure was administered 1 day after a 3-hr expo-

**Table 2.** Slopes from regressions of afternoon forced expiratory volume in 1 sec on ozone for six camp studies<sup>a</sup>

| Study                  | Slope (ml/ppb) | SE (slope) | p-value |
|------------------------|----------------|------------|---------|
| Fairview Lake, 1984    | -0.50          | 0.16       | 0.002   |
| Fairview Lake, 1988    | -1.29          | 0.27       | 0.0001  |
| Lake Couchiching, 1983 | -0.19          | 0.44       | 0.66    |
| CARES, 1986            | -0.29          | 0.10       | 0.003   |
| San Bernardino, 1987   | -0.84          | 0.20       | 0.0001  |
| Pine Springs, 1988     | -0.32          | 0.13       | 0.013   |

<sup>a</sup>For each study, data were analyzed in one model that fit subject-specific intercepts and one pooled  $O_3$  slope.

**Table 4.** Slopes of afternoon  $FEV_1$  and PEFR on ozone for all camp studies combined<sup>a</sup>

| Measurement | Slope            | SE (slope) | p-value |
|-------------|------------------|------------|---------|
| $FEV_1$     | -0.50 ml/ppb     | 0.07       | 0.0001  |
| PEFR        | +0.17 ml/sec/ppb | 0.33       | 0.62    |

Abbreviations:  $FEV_1$ , forced expiratory volume in 1 sec; PEFR, peak expiratory flow rate.

<sup>a</sup>For each study, data were analyzed in one model that fit subject-specific intercepts and one pooled  $O_3$  slope (no trend line).

**Table 3.** Slopes from regressions of afternoon peak expiratory flow rate on ozone for five camp studies<sup>a</sup>

| Study                  | Slope (ml/sec/ppb) | SE (slope) | p-value |
|------------------------|--------------------|------------|---------|
| Fairview Lake, 1984    | -2.00              | 0.80       | 0.013   |
| Lake Couchiching, 1983 | -2.66              | 1.32       | 0.046   |
| CARES, 1986            | -0.10              | 0.34       | 0.78    |
| San Bernardino, 1987   | -1.10              | 0.78       | 0.16    |
| Pine Springs, 1988     | +2.17              | 0.70       | 0.002   |

<sup>a</sup>For each study, data were analyzed in one model that fit subject-specific intercepts and one pooled  $O_3$  slope. Data not available for Fairview Lake, 1988.

**Table 5.** Slopes of afternoon  $FEV_1$  and PEFR on ozone, with trend terms included in the models; all camp studies combined<sup>a</sup>

| Measurement | $O_3$ slope      | SE (slope) | p-value |
|-------------|------------------|------------|---------|
| $FEV_1$     | -0.26 ml/ppb     | 0.07       | 0.0003  |
| PEFR        | -0.15 ml/sec/ppb | 0.34       | 0.65    |

Abbreviations:  $FEV_1$ , forced expiratory volume in 1 sec; PEFR, peak expiratory flow rate.

<sup>a</sup>For each study, data were analyzed in one model that fit subject-specific intercepts, slopes on one or more trend functions, and one pooled  $O_3$  slope.

sure to  $100 \mu\text{g}/\text{m}^3$   $\text{H}_2\text{SO}_4$  (17). Although the relevance of these data to the nonasthmatic subjects who experienced much lower acid levels at northeastern summer camps is not clear, they do demonstrate that potentiation can occur between these pollutants under laboratory conditions. Another recent chamber study investigating the interactive effects of  $\text{O}_3$  and  $\text{H}_2\text{SO}_4$  on lung function observed marginal evidence for interaction, but concluded that " $\text{O}_3$  is more important than  $\text{H}_2\text{SO}_4$  as a cause of short-term respiratory irritant effects" (18: p. 431). Further, the wide range of  $\text{FEV}_1/\text{O}_3$  slopes observed across the four northeastern camps (two in southern Ontario and two in New Jersey), which all experienced similar acid aerosol levels, suggests that differential acid exposures are not likely to be a significant factor in the inter-camp variation in response seen here.

Acute respiratory responses to  $\text{O}_3$  vary markedly across people, for reasons that are not entirely understood (5,19). This physiologic variation in responsiveness is surely present in the camp studies, but it is not likely to have a large effect on the average population response estimated for each camp. Differences in average population responsiveness might occur due to differing levels of prior exposure to  $\text{O}_3$ , with associated tolerance/adaptation. However, the results of our analysis do not suggest a systematically lower response in the California studies, where elevated prior exposures may have occurred.

Data have not been reported on comparative levels of airborne allergens during the camp studies. None of the subjects analyzed here reported a history of asthma, minimizing the likelihood of confounding by airborne allergens. However, given the lack of allergen data and the potential for substantial numbers of "silent hyperresponders" (8), this possibility cannot be completely discounted.

It is possible that several of the factors discussed above, acting together, could underlie the variation in  $\text{FEV}_1$  response observed across the six camps. However, given the many potential sources of camp-to-camp variability, it is both surprising and noteworthy that results are fairly consistent across the six studies. Further, a statistically significant drop in  $\text{FEV}_1$  was observed in the pooled data set in an analysis that incorporated both the within-camp and between-camp variability. Thus, in spite of variations across camps, it can be concluded that children exposed to  $\text{O}_3$  under natural conditions do experience acute decreases in lung function of the kind that have been demonstrated in great detail and precision in chamber studies. This

confirms the real-world public health significance of those laboratory observations and raises concern that other acute respiratory effects observed in chamber studies (e.g., pulmonary inflammation) may also occur in people exposed to ambient  $\text{O}_3$ .

Quantitative comparison of the population average  $\text{FEV}_1$  response observed here ( $-0.5 \text{ ml/ppb}$ ) with responses reported in chamber studies is complicated by the issues discussed above, as well as by differences in the designs and analytical methods used in the two types of studies. No in-depth analysis taking these differences into account has been reported to date. However, a brief review of chamber results suggests a reasonable degree of concordance. Setting aside nonlinearities in response, the results of the present study imply a 2.7% drop in  $\text{FEV}_1$  for a 120 ppb increase in  $\text{O}_3$  exposure in this population (mean  $\text{FEV}_1$  was 2.23 l in the data set analyzed). This change is nearly identical to the 2.8% change in  $\text{FEV}_1$  observed in 23 vigorously exercising male children exposed for 2.5 hr to 120 ppb  $\text{O}_3$  (1). The change is smaller than the 7–13% declines seen in studies of adults exposed for longer periods (6.6 hr) to between 80 and 120 ppb  $\text{O}_3$ , with intermittent exercise (2,20). In the absence of detailed further analysis, these data offer no evidence for systematic differences in the quantitative relationships between  $\text{FEV}_1$  and  $\text{O}_3$  observed in camp and chamber studies.

The data analysis presented here yields population-average results within and across camps. It does not explicitly address interindividual variations in responsiveness, which, as noted earlier, are likely to be present. As pointed out by Brunekreef and colleagues (19), this variation is difficult to analyze in detail in acute epidemiologic studies because it is obscured by substantial amounts of random variability. As a result, attempts to determine, for example, which individuals are most responsive to  $\text{O}_3$  using data from camp studies are problematic and generally should be avoided.

The pooled slope of  $\text{FEV}_1$  on  $\text{O}_3$  was reduced somewhat (in absolute magnitude) but remained statistically significant when the regression model included trend variables designed to account for training effects (14,15). A third-order polynomial provided a good fit to the observed time trend in  $\text{FEV}_1$ , which was characterized by a decline over the first four to five measurements, followed by a gradual increase and leveling. The pooled slope of  $\text{PEFR}$  on  $\text{O}_3$  increased slightly after accounting for a more simple linear increase in  $\text{PEFR}$  over time. The trend variables were highly statistically significant, confirming the impor-

tance of this phenomenon. These effects on the estimated associations between lung function and  $\text{O}_3$  suggest that confounding due to time trends may occur in some cases.

In summary, our results confirm a small, statistically significant, population-average decline in  $\text{FEV}_1$  (but not  $\text{PEFR}$ ) associated with  $\text{O}_3$  exposures that is qualitatively similar to that reported in chamber studies. Evidence for heterogeneity in average  $\text{FEV}_1$  response across studies was observed. Limitations of currently available data preclude definitive evaluation of the reasons for the variation in results across camps.

## REFERENCES

- McDonnell WF, Chapman RS, Leigh MW, Strope GL, Collier AM. Respiratory responses of vigorously exercising children to 0.12 ppm ozone exposure. *Am Rev Respir Dis* 132:875–879 (1985).
- Folinsbee LJ, McDonnell WF, Horstman DH. Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm ozone with moderate exercise. *J Air Pollut Control Assoc* 38:28–35 (1988).
- Spektor DM, Lippmann M, Liou PJ, Thurston GD, Citak K, James DJ, Bock N, Speizer FE, Hayes C. Effects of ambient ozone on respiratory function in active, normal children. *Am Rev Respir Dis* 137:313–320 (1988).
- Devlin RB, McDonnell WF, Mann R, Becker S, House DE, Schreinemachers D, Koren HS. Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung. *Am J Respir Cell Mol Biol* 4:72–81 (1991).
- McDonnell WF, Muller KE, Bromberg PA, Shy CM. Predictors of individual differences in acute response to ozone exposure. *Am Rev Respir Dis* 147:818–825 (1993).
- Higgins ITT, D'Arcy JB, Gibbons DI, Avol EL, Gross KB. Effects of exposures to ambient ozone on ventilatory lung function in children. *Am Rev Respir Dis* 141:1136–1146 (1990).
- Raizenne ME, Stern B, Burnett R, Spengler J. Acute respiratory function and transported air pollutants: observational studies (paper no. 87-32.6). Presented at the 80th annual meeting of the Air Pollution Control Association, New York, June 1987. Pittsburgh, PA:Air Pollution Control Association, 1987.
- Raizenne ME, Burnett RT, Stern B, Franklin CA, Spengler JD. Acute lung function responses to ambient acid aerosol exposures in children. *Environ Health Perspect* 79:179–185 (1989).
- Avol EL, Trim SC, Little DE, Spier CE, Smith MN, Peng R, Linn WS, Hackney JD, Gross KB, D'Arcy JB, Gibbons D, Higgins ITT. Ozone exposure and lung function in children attending a southern California summer camp (paper no. 90-150.3). Presented at the 83rd annual meeting and exhibition of the Air and Waste Management Association, Pittsburgh, Pennsylvania, June 1990. Pittsburgh, PA:Air and Waste Management Association, 1990.
- Avol EL, Trim SC, Little DE, Spier CE, Smith MN, Peng RC, Linn WS, Hackney JD. Ozone exposure and lung function: a southern California summer camp study. In: *Tropospheric ozone and the environment*:

- papers from an international conference (Berglund RL, Lawson DR, McKee DJ, eds), transaction series no. TR-19. Pittsburgh, PA: Air and Waste Management Association, 1991;90-99.
11. Spektor DM, Thurston GD, Mao J, He D, Hayes C, Lippmann M. Effects of single- and multiday ozone exposures on respiratory function in active normal children. *Environ Res* 55:107-122 (1991).
  12. Burnett R, Raizenne M, Krewski D. Acute health effects of transported air pollution: a study of children attending a residential summer camp. *Can J Stat* 18:365-367 (1990).
  13. ATS. American Thoracic Society Statement—Snowbird workshop on standardization of spirometry. *Am Rev Respir Dis* 119:831-838 (1979).
  14. Raizenne ME, Haines DA. Trends in repeated spirometry. *Am Rev Respir Dis* 141:A331 (1990).
  15. Hoek G, Brunekreef B. Time trends in repeated spirometry in children. *Eur Respir J* 5:553-559 (1992).
  16. Eschenbacher WL, Moore TB, Lorenzen TJ, Weg JG, Gross KB. Pulmonary responses of asthmatic and normal subjects to different temperature and humidity conditions in an environmental chamber. *Lung* 170:51-62 (1992).
  17. Frampton MW, Morrow PE, Cox C, Levy PC, Speers DM, Gibb FR, Condemi JJ, Utell MJ. Does pre-exposure to acidic aerosols alter airway responses to ozone in humans? *Am Rev Respir Dis* 145:A428 (1992).
  18. Linn WS, Shamoo DA, Anderson KR, Peng R, Avol EL, Hackney JD. Effects of prolonged, repeated exposure to ozone, sulfuric acid, and their combination in healthy and asthmatic volunteers. *Am J Respir Crit Care Med* 150:431-440 (1994).
  19. Brunekreef B, Kinney PL, Ware JH, Dockery D, Speizer FE, Spengler JD, Ferris BG Jr. Sensitive subgroups and normal variation in pulmonary function response to air pollution episodes. *Environ Health Perspect* 90:189-193 (1991).
  20. Horstman DH, Folinsbee LJ, Ives PJ, Abdulsalaam S, McDonnell WF. Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. *Am Rev Respir Dis* 142:1158-1163 (1990).

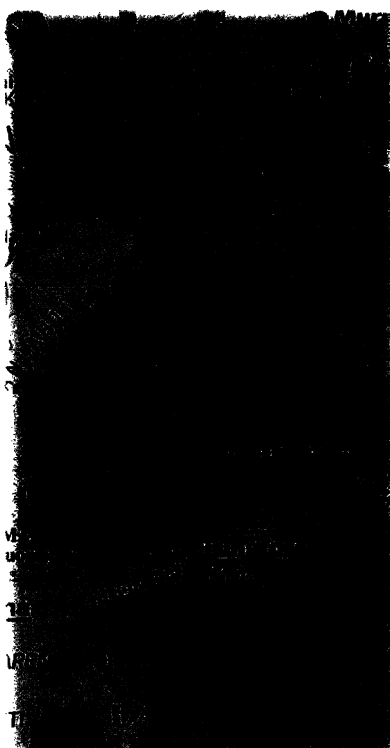
# Occupational Health and Safety in Progress

## Northern-Baltic-Karelian Regional Symposium

**Organized by the  
Finnish Institute  
of Occupational  
Health**

Main themes will include:

- Occupational Health Services
- Occupational and Environmental Hygiene and Toxicology
- Physiology, Ergonomics
- Safety and Risk Management
- Occupational Medicine
- Occupational Psychology



**August 12-14, 1996  
Lappeenranta, Finland**

**Deadline for Abstracts:  
April 30, 1996**

**Deadline for early  
registration:  
June 15, 1996**

For more information:  
Occupational Health and  
Safety in Progress  
c/o Finnish Institute of  
Occupational Health  
Symposium Secretariat,  
Anneli Vartio  
Topeliuksenkatu 41 a A  
FIN-00250 Helsinki  
FINLAND